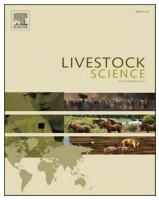
Livestock Science 254 (2021) 104763

Contents lists available at ScienceDirect

Livestock Science

journal homepage: www.elsevier.com/locate/livsci



The effect of the *SCD* genotype on litter size and weight at weaning

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HIGHLIGHTS

- The *SCD* SNP (rs80912566 T>C) does not impair overall sow performance.
- The *SCD*-T allele increases litter size at birth.
- The *SCD*-T allele decreases litter weight at weaning in primiparous young sows.
- *SCD*-T carrier sows produce milk with higher desaturation indices but less fat.
- Cautious management of *SCD*-TT lactating primiparous sows is recommended.

ARTICLE INFO

Keywords:
Genetic markers
Litter size
Milk
Pigs
Reproduction
Weaning weight

ABSTRACT

Genetic markers associated with meat quality may have adverse effects on reproduction performance, as shown for a variant in the leptin receptor (*LEPR*) gene. There is a single nucleotide polymorphism (SNP) in the stearoyl-CoA desaturase (*SCD*) gene that influences the monounsaturated fatty acid content of pork. Here, we investigate single and combined effects of the *SCD* (rs80912566 T>C) and *LEPR* (rs709596309 C>T) SNP on litter size and weaning weight. A total of 1,246 sows of known genotype for *SCD* (732 of which were also genotyped for *LEPR*) were monitored for age at farrowing, number of piglets born, born alive and weaned, and litter weight at weaning during two years. The effects of these SNP and of a variant in the diacylglycerol O-acyltransferase 2 (*DGAT2*) gene on milk fat content and fatty acid composition were also analysed in 99 primiparous sows. No adverse effects of the *SCD* SNP on overall reproductive performance were observed. However, in primiparous sows, we found that the *SCD*-T allele, the one that increases the monounsaturated fatty acid content, influenced negatively the weight of piglets at weaning (the piglets from *SCD*-TT sows weighted 126 g less at weaning than those from *SCD*-CT sows), especially in combination with the *LEPR*-TT genotype (the piglets from *SCD*-TT/*LEPR*-TT sows weighted 277 g less at weaning than piglets from *SCD*-C−/*LEPR*-C− sows). In contrast to *SCD*, the negative effect of the *LEPR*-TT sows on the piglets weaning weight was maintained in all parities (piglets from *LEPR*-TT sows were on average 126 g lighter than *LEPR*-C− sows). The unfavourable effect of the *SCD*-T allele on the litter weight of primiparous sows could be due to lower body reserves at first farrowing (*SCD*-TT pigs had 0.62 mm less backfat thickness at 205 d than *SCD*-CT pigs) together with larger litters at birth (the *SCD*-TT sows had 0.32 more piglets born alive per litter than the *SCD*-CT sows). The milk of primiparous sows with the *SCD*-T allele showed higher desaturation indices (i.e. higher monounsaturated to saturated fatty acid ratios), but lower fat content. No relevant effects of the *DGAT2* SNP on milk traits were found. In conclusion, the *SCD* SNP does not impact overall reproductive performance in pigs, but cautious management of *SCD*-TT gilts is recommended.

1. Introduction

Sire lines used in crossbreeding schemes and purebred specialized lines for premium markets are often selected for meat quality traits. Since these traits are expensive to measure and cannot be recorded in vivo, the prediction of their genetic value is based on some form of

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https://doi.org/10.1016/j.livsci.2021.104763
Received 2 August 2021; Received in revised form 15 October 2021; Accepted 2 November 2021
Available online 6 November 2021

marker-assisted or weighted genomic selection (Pena et al., 2016). One of these markers is a recessive missense mutation in the leptin receptor (*LEPR*) gene (Óvilo et al., 2005) that increases intramuscular fat content and saturated fatty acids and decreases polyunsaturated saturated fatty acids (Ros-Freixedes et al., 2016; Suárez-Mesa et al., 2021). We have recently shown that this *LEPR* polymorphism is also at the origin of a maternal effect that influences body weight, particularly at weaning (Solé et al., 2021a). Maternal effects, which include milk yield and other mothering abilities, are the main limiting genetic factor for improving piglet survival during the nursing period (Nguyen et al., 2021). Thus, the number and weight of weaned piglets per sow are crucial for reproductive success and, even for a sire line, reproductive success is important issue for the overall line efficiency.

Another genetic marker with a major effect on fat quality in pigs is a polymorphism in the promoter of the steatoryl-CoA desaturase (*SCD*) gene (Estany et al., 2014), which influences the monounsaturated fatty acids (MUFA) content in the adipose tissue. The SCD is the rate-limiting enzyme required for the biosynthesis of MUFA, catalysing in particular the desaturation of palmitic acid (C16:0) and stearic acid (C18:0) to palmitoleic acid (C16:1n-7) and oleic acid (C18:1n-9), respectively. In connection with *SCD*, there is an exonic polymorphism in the diacylglycerol O-acyltransferase 2 (*DGAT2*) gene that specifically impacts C16:1n-7 (Solé et al., 2021b). The DGAT2 is the enzyme that, together with DGAT1 and SCD, intervenes in the final step of the biosynthesis of triglycerides. Although several studies in cattle associate *SCD* with milk fatty acid composition (Li et al., 2016; Gu et al., 2019) and *DGAT1* with milk yield and composition (Grisart et al., 2002; Tabaran et al., 2015), to our knowledge similar effects have not been investigated in pigs yet. The aim of this study was to evaluate potential side-effects of *SCD* on sow productivity and of *SCD* and *DGAT2* on milk fat content and fatty acid composition.

2. Material and methods

2.1. Animals and phenotypes

All pigs used in the study were raised and slaughtered in commercial units following applicable regulations and good practice guidelines on the protection of animals kept for farming purposes, during transport and slaughter. Reproductive data were obtained from a Duroc nucleus farm managed using standard practices, where gilts were monitored for oestrus at around 6.5 months of age and then bred on their second detected oestrus. Sows were rebred on their first oestrus after weaning. The performance of 4121 parities from 1246 sows was monitored during three and a half years (15 contemporary year-season farrowing batches). Age at farrowing, number of piglets born alive, number of weaned piglets and litter weight at weaning (23 days, 2 SD) was recorded. In line with commercial practice, litter size was equalized by cross-fostering within 24 h of birth and creep feed was offered to litters from about 10 days after birth until weaning. In a random set of 99 primiparous sows from six batches, a 15-mL sample of milk was extracted at around the end of the first week of lactation (4.2 days, 1.6 SD) from anterior teats following intramuscular oxytocin injection (20 UI; Hormonipra, Spain). Milk samples were stored at −40 °C until analysis. Milk fat content and fatty acid composition were determined in duplicate using the gravimetric solvent method of Hara and Radin (1978) as adapted by Feng et al. (2004) followed by quantitative gas chromatography (Bosch et al., 2009). The amount of each fatty acid was expressed as the percentage of each individual fatty acid relative to total fatty acids. The proportion of saturated (SFA: C10:0, C12:0, C14:0, C16:0, C18:0, C20:0), monounsaturated (MUFA: C14:1n-5, C16:1n-7, C18:1n-7, C18:1n-9, C20:1n-9) and polyunsaturated (PUFA: C18:2n-6, C18:3n-3, C18:3n-6, C20:2n-6) fatty acids were calculated. Complementarily, 1070 barrows were raised in 15 batches under commercial conditions (Solé et al., 2021b) until 30 weeks of age (207 days, SD 8), at which time they were weighted and their backfat and loin thickness were ultrasonically measured at 5 cm off the midline at the position of the last rib (Renco, Minneapolis, USA). A few days later (211 days, 9 SD), all pigs in a batch were slaughtered at a time in an abattoir equipped with a carbon dioxide stunning system, where the carcass backfat and loin thickness at 6 cm off the midline between the third and fourth last ribs were predicted with an automatic carcass grading equipment (Auto-FOM, Frontmatec Group, Denmark).

2.2. Genotyping

All sows and barrows used in the experiments were genotyped for the *SCD* (rs80912566 T>C, SSC14) single nucleotide polymorphism (SNP). A subset of 732 sows plus all the barrows were also genotyped for the *LEPR* (rs709596309 C>T, SSC6) SNP. Sows with milk records were additionally genotyped for *DGAT2* (ss7315407085 G>A, SSC9). Genomic DNA was isolated from blood and ear notches (sows) and muscle (barrows) using standard protocols. Quantification and purity of DNA was assessed by spectrophotometry with a NanoDrop N-1000 Spectrophotometer (Thermo Fisher Scientific, Waltham, MA, USA) and the integrity was tested by electrophoresis in agarose gels. The three polymorphisms were genotyped by High-Resolution Melt analysis (Luminaris Colour HRM Master Mix, Thermo Scientific) in a real-time thermocycler (QuantStudio3, Applied Biosystems) using 20 ng of genomic DNA, 0.4 μM of each primer in final volume of 5 μL. Thermocycling conditions were 50 °C 2 min, 95 °C 10 min, and 40 cycles of 95 °C 15 s, 60 °C 1 min, followed by a high-resolution melting curve starting with a denaturation at 95 °C for 15 s, annealing at 60 °C for 1 min and a slow ramp at 0.015 °C/*sec* up to 95 °C. High Resolution Melt software v3.1 (Applied Biosystems, Thermo Fisher Scientific, Waltham, MA, USA) was used for the melting data analysis and the genotyping of the samples. Primers used to genotype the *LEPR* rs709596309 and *DGAT2* ss7315407085 polymorphisms are described in Ros-Freixedes et al. (2016) and Solé et al. (2021b), respectively. The *SCD* rs80912566 SNP was genotyped with primers 5′-AGCGAATAAAAGGGGTCAGAGG-3′ and 5′-TTAAAGGCTAGAGCT GGCAGTG-3′, which amplify a 64 bp-long amplicon.

2.3. Statistical analyses

The effect of the *SCD* genotype on reproduction traits (number of total piglets born, born alive and weaned, and litter and average piglet weight at weaning) was estimated using a repeatability model that accounted for the effect of the sow and with the sow *SCD* (TT, CT and CC) and *LEPR* genotypes (TT and C−, which includes CC and CT), the parity number (from 1 to 8) and the batch (15 batches) as systematic effects. The duration of lactation was included as a covariate for weaning weight. In matrix notation, the model was $\boldsymbol{y} = \mathbf{Xb} + \mathbf{Zs} + \mathbf{e}$, where **y** is the vector of observations for a trait; **b, s** and **e** are the vectors of systematic, sow and residual effects, respectively; and **X** and **Z** are the incidence matrices that relate **b** and **s** with **y**, respectively. The traits were assumed to be conditionally normally distributed as $[\mathbf{y} \mid \mathbf{b}, \mathbf{s}, \mathbf{I}\sigma_e^2]$ ~x223C N $(\mathbf{Xb} + \mathbf{Zs}, \mathbf{I}\sigma_e^2)$, where σ_e^2 is the residual variance and **I** the appropriate identity matrix. The sow effects conditional on the sow variance σ_s^2 were assumed multivariate normally distributed with mean zero and variance $\mathbf{I}\sigma_s^2$. The sow and the parity number were removed from the model when only data on first parities were used. A simple animal model was used for milk (fat content and fatty acid composition) and production (body weight and backfat and loin thickness) traits. The model for milk traits included the batch (6 batches), the sow *SCD, LEPR* and *DGAT2* (GG, AG and AA) genotypes and the duration of lactation and the milk fat content (for fatty acids) as covariates, whereas the model for production traits included the batch (15 batches), the pig *SCD* and *LEPR* genotype and the age at measurement as a covariate.

Genotype effects were estimated in a Bayesian setting with the TM software (Legarra et al., 2008; http://genoweb.toulouse.inra.fr/~x223Calegarra/tm_folder [deposited: 3 August 2011]). Statistical inferences

for each of the above models were derived from the samples of the marginal posterior distribution using a Gibbs sampling Markov chain Monte Carlo algorithm with a chain of 500,000 iterations, where the first 100,000 were discarded and one sample out of 100 iterations retained. Flat priors were used for **b** and variance components. Convergence was tested using the Z-criterion of Geweke (1992) and visual inspection of convergence plots. Statistical evidence for the effects of the *LEPR* polymorphism was calculated as the marginal posterior probability of the difference between genotype estimates being greater ($P_{[>0]}$) or lower ($P_{[<0]}$) than zero. We considered that there was strong (suggestive) evidence of difference between the genotypes when the probability of that difference being greater or lower than zero was of at least 0.95 (0.90).

3. Results and discussion

The *SCD* SNP did not have a negative impact on litter size and weight at weaning, rather the opposite (Table 1). Using all parities, the results indicate that the *SCD*-T allele, the one that enhances MUFA, increased litter size at birth (*SCD*-TT sows had 0.33 more piglets born alive per litter than *SCD*-CT sows, $P_{[>0]}$=0.99). The *SCD*-T allele also decreased the number of stillborn piglets (*SCD*-TT sows had 0.16 less stillborn piglets per litter than *SCD*-CT sows, $P_{[<0]}$=0.98), with no effect on the weight of piglets at weaning. A similar trend was observed if litter size was not adjusted for the *LEPR* SNP, thereby using all sows genotyped for the *SCD* SNP (593, 603 and 50 sows for *SCD*-TT, CT and CC, respectively, accounting for 4121 parities). With this set of data, *SCD*-TT sows had 0.14 more piglets born alive ($P_{[>0]}$=0.92) and 0.09 less stillborn piglets ($P_{[<0]}$=0.95) per litter. Moreover, *SCD* genotypes did not differ for the age at the first farrowing and reproduction rate (the average number of parities in two years was 3.97, for *SCD*-CT sows, and 3.85, for *SCD*-TT sows). Taken as a whole, these results would confirm that the *SCD* SNP is not expected to entail adverse consequences in the reproductive lifetime of a breeding sow. This contrasts with the negative effects of *LEPR*-T on reproduction of homozygous sows. In line with previous findings (Solé et al., 2021a), results obtained here indicate that *LEPR*-TT sows, as compared with *LEPR*-C− sows, showed a delay in the age at first farrowing (5.7 days, $P_{[>0]}$=0.99) and a reduction in the litter size at birth (−0.23 piglets born alive per litter, $P_{[<0]}$=0.94). To our knowledge, no studies so far have investigated the effects of *SCD* on reproduction in pigs. However, recent research in cattle indicates that enhanced *SCD* activity can be beneficial for oocyte development. Thus, Aardema et al. (2017) observed that the desaturation of C18:0 into C18:1n-9 via SCD in cumulus cells would protect the oocyte against toxicity by saturated free fatty acids. On the other hand, Asadollahpour et al. (2014) reported a preliminary evidence that a SNP in the bovine *SCD* gene could be associated with pregnancy length, dry days and open days.

The differences between *SCD* genotypes for litter size in the first farrowing were in line with the results obtained using all parities (Table 1). However, a further look at the effects in the first farrowing reveals that the *SCD* SNP is not neutral with respect to weaning weight. In particular, piglets from the first farrowing of *SCD*-TT sows weighted 126 g less at weaning ($P_{[<0]}$=0.97) than those from *SCD*-CT sows. The negative impact of the *SCD*-TT sows on weaning weight was maintained after removing the *LEPR* genotype from the model (−137 g, $P_{[<0]}$=0.98). This effect could be attributed to differences in litter size at birth, but not exclusively. First, because *SCD*-TT sows only weaned lighter piglets at first parity while they had larger litters in all their parities and, second, because it was still detected after adjusting for the number of total piglets born and age at farrowing. Moreover, as litter size was equalized during lactation (Table 1), the effect of increased litter size on piglet weight at weaning reduced to 22 g less per additional piglet born ($P_{[<0]}$ >0.99). Cross-fostering was done regardless of the genotype, so it is most likely that more piglets from *SCD*-TT sows, with larger litters at birth, have been transferred to *SCD*-CT litters than vice versa. If so, the negative effect of primiparous *SCD*-TT sows on weaning weight could have been even greater than the estimated here.

The negative effect of primiparous *SCD*-TT sows on weaning weight, although smaller in magnitude, was also detected using all available sows, i.e. also including those that were genotyped for *SCD* but not for *LEPR* (piglets from *SCD*-TT sows weighted 89 g less at weaning than piglets from *SCD*-CT sows ($P_{[<0]}$=0.98). Contrarily, it vanished when only the litters that were not genotyped for *LEPR* (205 from *SCD*-TT sows and 182 *SCD*-CT sows) were analysed (−10 g, $P_{[<0]}$=0.57). This finding, given that most of these litters were from the earliest five batches, suggests that *SCD*-TT gilts in these batches were favoured by some temporary effect. Interestingly, the average age at first farrowing in the first five batches occurred 19 days later than in the other of batches (396 days vs 377 days), which in practice could mean that skipping a heat may be beneficial for the *SCD*-TT gilts. All in all, these results indicate that, despite the lack of unfavourable effects of the *SCD* SNP over the sow lifetime performance, caution should be taken in the management of *SCD*-T carrier gilts, particularly if the *SCD*-TT genotype combines with the *LEPR*-TT genotype (Fig.1). Findings here confirm that *LEPR*-TT sows produce lighter pigs than *LEPR*-C− sows, both in the first (−154 g, $P_{[<0]}$>0.99) and in all parities (−126 g, $P_{[<0]}$>0.01). They also show that the effect of the *SCD* SNP is independent of the *LEPR* genotype. The detrimental effect of primiparous *SCD*-TT sows on weaning weight as compared to that of *SCD*-C− sows (CT and CC sows were included in a single group due to the limited number of CC sows) was maintained between *LEPR* genotypes (−125 g, $P_{[<0]}$=0.87, if *LEPR* is TT, and −126 g, $P_{[<0]}$=0.96, if *LEPR* is C−, Fig. 1). As a result, the effect of one of the genotypes adds to the other and thus the piglets in litters from primiparous *SCD*-TT/*LEPR*-TT sows weighted 277 g less at weaning ($P_{[<0]}$>0.99) than piglets in litters from *SCD*-C−/*LEPR*-C− sows.

Table 1
Mean (standard deviation) of the marginal posterior distribution of the difference between *SCD* and *LEPR* genotypes for litter size and weight in all parities and in the first parity.

Trait	*SCD*			*LEPR*
	Mean $_{TT}$	Mean $_{TT-CT}$	Mean $_{TT-CC}$	Mean $_{TT-C-}$
All parities				
No. of sows	331 (TT)	357 (CT)	44 (CC)	
No. of parities	904	1101	135	
No. of total piglets born	12.1	0.25 (0.15) [b]	0.20 (0.30)	−0.21 (0.15) [c]
No. of piglets born alive	11.4	0.33 (0.14) [a]	0.26 (0.28)	−0.23 (0.14) [c]
No. of piglets stillborn	0.7	−0.16 (0.08) [b]	−0.08 (0.16)	−0.10 (0.08)
No. of weaned piglets	9.6	0.00 (0.07)	0.07 (0.15)	−0.07 (0.08)
Litter weight at weaning, kg	52.3	−0.16 (0.57)	−0.08 (1.20)	−1.73 (0.59) [a]
Piglet weight at weaning, g	5430	−5 (41)	−23 (85)	−126 (43) [a]
First parity				
No. of sows	290 (TT)	298 (CT)	34 (CC)	
Age at farrowing, days	376.8	1.2 (1.7)	−2.9 (3.7)	5.7 (1.8) [a]
No. of total piglets born	11.3	0.21 (0.20)	0.66 (0.43) [b]	0.02 (0.21)
No. of piglets born alive	10.6	0.23 (0.20)	0.74 (0.43) [b]	−0.08 (0.21)
No. of piglets stillborn	0.6	−0.18 (0.16)	0.09 (0.28)	−0.18 (0.15)
No. of weaned piglets	9.5	−0.00 (0.12)	0.22 (0.26)	−0.07 (0.13)
Litter weight at weaning, kg	44.8	−1.30 (0.82) [c]	−0.38 (1.78)	−2.02 (0.85) [a]
Piglet weight at weaning, g	4761	−126 (63) [c]	−128 (137)	−155 (65) [a]

[a] ,b,c Posterior probability of the difference between genotypes being greater (if positive) or lower (if negative) than zero is 0.99 ([a]), 0.95 ([b]) and 0.90 ([c]).

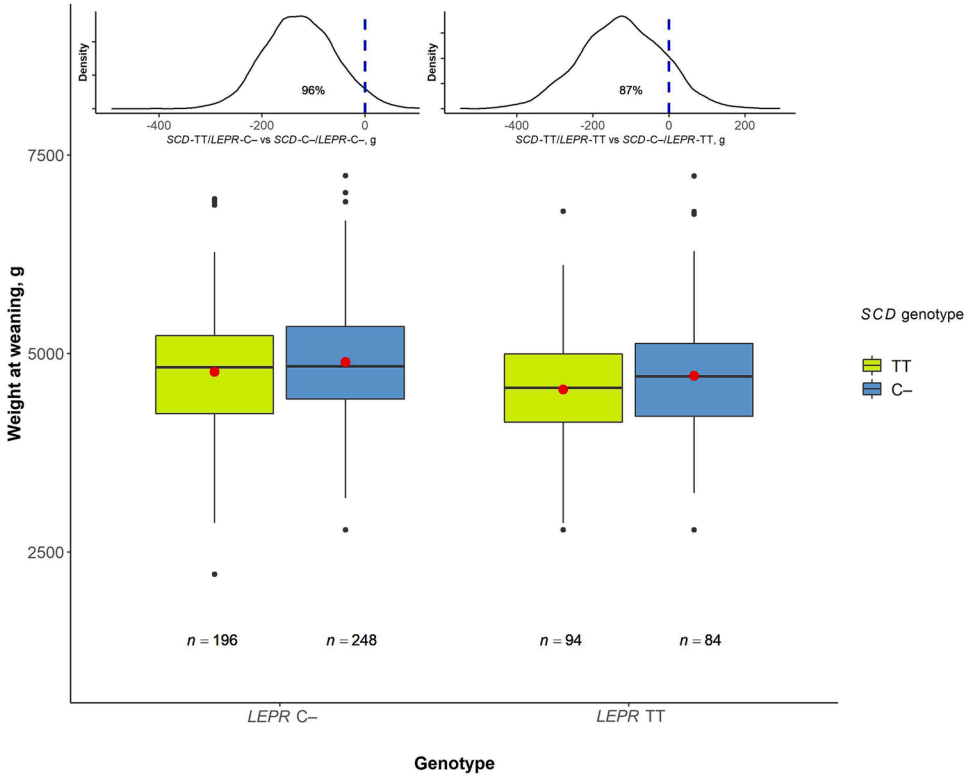

Fig. 1. Boxplot distribution of weight at weaning at first farrowing by *SCD* and *LEPR* genotype, with the red point indicating the mean value. Values represented are adjusted for systematic effects. The marginal posterior distribution of the difference between genotypes is depicted on the top of each panel, with the blue dotted line indicating the zero value (no difference) and the accompanying percentage standing for the posterior probability of the difference being lower (area under of the curve at the left side of the line) than zero. Sample size (*n*) is given below each boxplot.

We have seen that the unfavourable effect of *LEPR*-TT sows on weaning weight can be attributed to a lower capacity of these sows to mobilize body reserves (Solé et al., 2021a), which may lead to impaired milk production. However, this does not seem the case for the *SCD*-T allele, whose potential negative effect on weaning weight is restricted to the first farrowing. Moreover, there is no evidence that the *SCD* genotype has a relevant impact on circulating free fatty acids after fasting (Tor et al., 2021), the source from which energy reserves are mobilized to organs and tissues. Alternatively, we have investigated whether this effect could be explained by either body condition at puberty or milk quality. From examining production data, we found that *SCD*-TT pigs presented lower body condition at the end of fattening (Table 2), with 0.62 mm less backfat thickness ($P_{[<0]}$=0.96) and 0.82 mm less loin thickness ($P_{[<0]}$=0.98) than contemporaneous *SCD*-CT pigs. Also, although only using data from 28 *SCD*-TT and 35 *SCD*-C– barrows that were individually monitored using automatic feeders, we found evidence that in this period the *SCD*-TT pigs consumed less feed than the *SCD*-C– pigs (18.6 kg less from 70 d to 200 d of age, $P_{[<0]}$= 0.99). On the other hand, minor changes in milk content and composition were observed in lactating primiparous sows (Table 3). Thus, as compared to *SCD*-C– sows, the milk of *SCD*-TT sows tended to have less fat (0.73%, $P_{[>0]}$=0.10) but more MUFA, especially for the C18:1n-9/C18:0 ratio, which increased with the T allele content (Table 3). In contrast, neither the *DGAT2* (Table 3) nor the *LEPR* (Solé et al., 2021a) SNP substantially altered milk characteristics. In particular, we did not observe, as in muscle, that the milk of *DGAT2-GG* sows was richer in C16:1n-7 (*DGAT2*-GG sows had 0.05% less C16:1n-7 than *DGAT2*-AA sows, $P_{[<0]}$=0.47) nor had higher values of the ratio of C16:1n-7 to C16:0 (the value of the C16:1n-7 to C16:0 ratio was 0.06 (x10) lower in *DGAT2*-GG sows than in *DGAT2*-AA sows, $P_{[<0]}$=0.37). Milk fatty acid profile tends to reflect the maternal diet composition during gestation (Laws et al., 2009). However, fatty acids in milk also originate from de novo synthesis in the mammary gland or, particularly in case of negative energy balance, from fatty acids released from body fat stores. The fact that the effect of the *SCD*-T allele as a desaturation enhancer can be detected in milk could be interpreted as an indirect evidence that dietary fatty acids are less prevalent in the milk of sows carrying this allele. Thus, while differences between *SCD* genotypes for milk fat content could be

Table 2
Mean (standard deviation) of the marginal posterior distribution of the difference between *SCD* genotypes for live and carcass weight and fatness.

Trait	*SCD* genotype		
	Mean $_{TT}$	Mean $_{TT-CT}$	Mean $_{TT-CC}$
Live measurements (207 d of age)			
No. of pigs	184 (TT)	447 (CT)	254 (CC)
Body weight, kg	123.7	−1.01 (0.98)	−0.65 (1.11)
Backfat thickness, mm	21.2	−0.62 (0.36) [b]	−0.27 (0.41)
Loin thickness, mm	47.2	−0.82 (0.41) [b]	−1.01 (0.46) [a]
Carcass measurements (211 d of age)			
No. of pigs	253 (TT)	532 (CT)	285 (CC)
Carcass weight, kg	95.3	−0.87 (0.74)	−0.57 (0.85)
Backfat thickness, mm	26.1	−0.16 (0.35)	0.38 (0.40)
Loin thickness, mm	44.4	−0.25 (0.60)	−1.30 (0.68)

[a] ,b Posterior probability of the difference between genotypes being greater (if positive) or lower (if negative) than zero is 0.99 ([a]) and 0.95 ([b]).

Table 3
Mean (standard deviation) of the marginal posterior distribution of the difference between *SCD* and *DGAT2* genotypes for fat content and fatty acid composition in milk.

Trait	*SCD* genotype			*DGAT2* genotype	
	Mean TT	Mean TT-CT	Mean TT-CC	Mean GG-GA	Mean GG-AA
No. of sows	39 (TT)	50 (CT)	10 (CC)	50 (GA)	18 (AA)
Milk fat,% DM[A]	6.82	−0.63 (0.59)	−1.50 (0.91) [b]	−0.15 (0.50)	−0.32 (0.63)
Fatty acid,% FA[B]					
C14:0	3.1	0.14 (0.19)	−0.15 (0.29)	−0.11 (0.17)	0.05 (0.21)
C16:0	26.7	0.11 (0.75)	−1.31 (1.16)	0.20 (0.67)	0.22 (0.84)
C18:0	4.3	−0.28 (0.22) [c]	−0.36 (0.33)	0.20 (0.19)	0.10 (0.23)
SFA	34.9	−0.08 (0.86)	−1.91 (1.32) [c]	0.24 (0.74)	0.36 (0.92)
C16:1n-7	8.5	1.21 (0.67) [b]	0.21 (1.03)	−0.05 (0.57)	−0.05 (0.71)
C18:1n-9	35.4	−0.75 (1.32)	3.28 (2.05) [b]	−0.13 (1.19)	1.03 (1.48)
C18:1n-7	2.7	0.17 (0.09) [b]	0.27 (0.14) [b]	0.06 (0.08)	0.13 (0.10)[c]
MUFA	47.5	0.44 (1.05)	3.61 (1.63) [a]	−0.14 (0.89)	1.29 (1.10)
C18:2n-6	15.5	−0.34 (0.75)	−1.41 (1.17)	−0.12 (0.66)	−1.53 (0.81)[b]
C18:3n-3	1.1	−0.02 (0.06)	−0.11 (0.09)	−0.01 (0.05)	−0.12 (0.06)[b]
PUFA	17.7	−0.42 (0.83)	−1.73 (1.29) [c]	−0.10 (0.72)	−1.74 (0.87)[b]
Fatty acid ratio					
C16:1n-7/C16:0 (x10)	3.1	0.42 (0.19) [a]	0.17 (0.29)	−0.06 (0.17)	−0.06 (0.21)
C18:1n-9/C18:0	8.5	0.46 (0.35) [c]	1.49 (0.54) [a]	−0.39 (0.30)	0.02 (0.37)
(C16:1n-7 + C18:1n-7) /C16:0 (x10)	4.2	0.42 (0.17) [a]	0.28 (0.26)	−0.03 (0.15)	0.05 (0.19)
MUFA/SFA	1.4	0.01 (0.06)	0.19 (0.09) [b]	0.00 (0.05)	0.04 (0.06)

[A] DM: dry matter;.

[B] SFA: saturated fatty acids (C10:0 + C12:0 + C14:0 + C16:0 + C18:0 + C20:0); MUFA: monounsaturated fatty acids (C14:1n-5 + C16:1n-7 + C18:1n-9 + C18:1n-7 + C20:1n-9); PUFA: polyunsaturated fatty acids (C18:2n-6 + C18:3n-3 + C18:3n-6 + C20:2n-6). Only fatty acids with a percentage greater than 1% are given.

[a] ,b,c Posterior probability of the difference between genotypes being greater (if positive) or lower (if negative) than zero is 0.99 ([a]), 0.95 ([b]) and 0.90 ([c]).

attributed to differential accumulation of body reserves prior to the first lactation, the differences in milk fatty acid composition can be explained by a differential desaturase activity in the mammary gland, in line with results in dairy cows, where the *SCD* gene has been associated with the MUFA content in milk (Mele et al., 2007; Li et al., 2016).

4. Conclusion

Despite the lack of relevant effects of the *SCD* genotype on reproduction traits, caution should be taken on the influence of the *SCD*-T allele on the weight of the piglets at weaning in the first farrowing, particularly if the *LEPR*-T allele is also segregating in the population. Causes behind this effect still require clarification, but the association of the *SCD*-T allele with delayed body growth and larger litters can be a plausible hypothesis.

Funding

This research was supported by the Spanish Ministry of Science, Innovation & Universities and the EU Regional Development Funds (grant RTI2018–101,346-B-I00). ES is recipient of a PhD scholarship from the University of Lleida.

Authorship statement

ES: Formal analysis, Investigation, Writing - original draft. RRF: Data curation, Funding acquisition, Writing - review & editing. LB: Investigation. MT: Methodology. SG: Investigation. RNP: Methodology, Writing - review & editing. JR: Conceptualization, Data curation, Resources. JE: Conceptualization, Funding acquisition, Writing - original draft.

Declaration of Competing Interest

Authors JE, MT, and RNP are named on the patent EP2902498B1-filed by University of Lleida on the use of markers of *SCD* for marker-assisted selective breeding in pigs.

Acknowledgements

We acknowledge the personnel at Selección Batallé for their cooperation for the recording of on-farm data and sample collection. We gratefully acknowledge Pilar Sopeña from the Animal Breeding group, University of Lleida, for laboratory assistance.

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